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L16 ANSWER 1 OF 5 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:234810 HCAPLUS
TITLE: Analytical performance of a sandwich enzyme immunoassay for pre.beta.1-HDL in stabilized plasma
AUTHOR(S): Miida, Takashi; **Miyazaki, Osamu**; Nakamura, Yasushi; Hirayama, Satoshi; Hanyu, Osamu; **Fukamachi, Isamu**; Okada, Masahiko
CORPORATE SOURCE: Division of Clinical Preventive Medicine, Department of Community Preventive Medicine, Niigata University Graduate School of Medical and Dental Sciences, Niigata, 951-8510, Japan
SOURCE: Journal of Lipid Research (2003), 44(3), 645-650
CODEN: JLPRAW; ISSN: 0022-2275
PUBLISHER: Lipid Research, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have established an immunoassay for pre.beta.1-HDL (the initial acceptor of cellular cholesterol) using a monoclonal antibody, MAb55201. Because pre.beta.1-HDL is unstable during storage, fresh plasma must be used for pre.beta.1-HDL measurements. In this study, we describe a method of stabilizing pre.beta.1-HDL, and evaluate the anal. performance of the immunoassay for pre.beta.1-HDL. Fresh plasma was stored under various conditions with or without a pretreatment consisting of a 21-fold diln. into 50% (vol./vol.) sucrose. Pre.beta.1-HDL concn. was measured by immunoassay. In nonpretreated samples, pre.beta.1-HDL decreased significantly from the baseline after 6 h at room temp. Although pre.beta.1-HDL was more stable at 0.degree.C than at room temp., it increased from 30.2 .+-. 8.5 (SE) to 56.5 .+-. 5.5 mg/l apolipoprotein A-I (apoA-I) ($P < 0.001$) in hyperlipidemics, and from 18.4 .+-. 1.2 to 37.9 .+-. 3.3 mg/l apoA-I ($P < 0.001$) in normolipidemics after 5-day storage. After 30-day storage at -80.degree.C, pre.beta.1-HDL increased from 29.0 .+-. 4.0 to 38.0 .+-. 5.7 mg/l apoA-I ($P < 0.001$) in hyperlipidemics, whereas it did not change in normolipidemics. In pretreated samples, pre.beta.1-HDL concn. did not change significantly under any of the above conditions. Moreover, pre.beta.1-HDL concns. detd. by immunoassay correlated with those detd. by native two-dimensional gel electrophoresis ($n = 24$, $r = 0.833$, $P < 0.05$). An immunoassay using MAb55201 with pretreated plasma is useful for clin. measurement of pre.beta.1-HDL.
CC 9 (Biochemical Methods)
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:126040 HCAPLUS
DOCUMENT NUMBER: 138:366757
TITLE: LCAT-Dependent Conversion of Pre.beta.1-HDL into .alpha.-Migrating HDL is Severely Delayed in Hemodialysis Patients
AUTHOR(S): Miida, Takashi; **Miyazaki, Osamu**; Hanyu, Osamu; Nakamura, Yuichi; Hirayama, Satoshi; Narita, Ichiei; Gejyo, Fumitake; Ei, Isei; Tasaki, Kazuyuki; Kohda, Yutaka; Ohta, Takashi; Yata, Syogo; **Fukamachi, Isamu**; Okada, Masahiko
CORPORATE SOURCE: Department of Community Preventive Medicine, Division of Clinical Preventive Medicine, Niigata University, Graduate School of Medical and Dental Sciences, Niigata, Japan
SOURCE: Journal of the American Society of Nephrology (2003),

14(3), 732-738
CODEN: JASNEU; ISSN: 1046-6673
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Pre.beta.1-HDL is a minor HDL subfraction that acts as an efficient initial acceptor of cell-derived free cholesterol. During 37.degree. incubation, plasma pre.beta.1-HDL decreases over time due to its conversion to .alpha.-migrating HDL by lecithin:cholesterol acyltransferase (LCAT). This conversion may be delayed in hemodialysis patients who have decreased LCAT activity. To clarify whether LCAT-dependent conversion of pre.beta.1-HDL to .alpha.-migrating HDL is delayed in hemodialysis patients, pre.beta.1-HDL concns. were detd. in 45 hemodialysis patients and 45 gender-matched control subjects before and after 37.degree. incubation with and without the LCAT inhibitor. It was found that the baseline pre.beta.1-HDL concn. in hemodialysis patients was more than twice that in the controls (44.9.+-.21.4 vs. 19.8.+-.6.7 mg/L apoAI). After 2-h incubation, the LCAT-dependent decrease in pre.beta.1-HDL in hemodialysis patients was about one-third of that in the controls (30.+-.27 vs. 97.+-.17% of baseline). The LCAT-dependent rate of decrease in pre.beta.1-HDL levels (DRpre.beta.1) was the same for samples from hemodialysis patients exhibiting normal (.gtoreq.1.03 mM) and low HDL-cholesterol levels (32.+-.32 vs. 28.+-.23% of baseline; NS). DRpre.beta.1 was pos. correlated with LCAT activity. In conclusion, the LCAT-dependent conversion of pre.beta.1-HDL to .alpha.-migrating HDL is severely delayed in hemodialysis patients. The impaired catabolism of pre.beta.1-HDL may accelerate atherosclerosis in hemodialysis patients.

CC 14-5 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 63
ST LCAT prebeta1 HDL hemodialysis atherosclerosis
IT Atherosclerosis
Human
(LCAT-dependent conversion of Pre.beta.1-HDL into .alpha.-migrating HDL is severely delayed in hemodialysis patients)
IT Dialysis
(hemodialysis; LCAT-dependent conversion of Pre.beta.1-HDL into .alpha.-migrating HDL is severely delayed in hemodialysis patients)
IT Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(high-d.; LCAT-dependent conversion of Pre.beta.1-HDL into .alpha.-migrating HDL is severely delayed in hemodialysis patients)
IT 9031-14-5, Lecithin-cholesterol acyltransferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(LCAT-dependent conversion of Pre.beta.1-HDL into .alpha.-migrating HDL is severely delayed in hemodialysis patients)
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 2002:706782 HCAPLUS
DOCUMENT NUMBER: 138:35657
TITLE: Delineation of the role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL
AUTHOR(S): Sviridov, Dmitri; **Miyazaki, Osamu**; Theodore, Kally; Hoang, Anh; **Fukamachi, Isamu**; Nestel, Paul
CORPORATE SOURCE: Baker Medical Research Institute, Melbourne, Australia
SOURCE: Arteriosclerosis, Thrombosis, and Vascular Biology (2002), 22(9), 1482-1488
CODEN: ATVBFA; ISSN: 1079-5642

PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Objective - The role of pre-.beta.1-high d. lipoprotein (pre-.beta.1-HDL) in cholesterol efflux was investigated by sepg. human plasma into purified pre-.beta.1-HDL and pre-.beta.1-HDL-deficient plasma by using a monoclonal antibody specifically reacting with pre-.beta.1-HDL. Methods and Results - When compared with whole plasma, pre-.beta.1-HDL-deficient plasma was equally efficient in promoting cholesterol efflux from human skin fibroblasts and THP-1 human macrophage cells. When added at the same apolipoprotein A-I concn., pre-.beta.1-HDL was less effective than whole plasma to promoting cholesterol efflux from fibroblasts but equally effective in promoting cholesterol efflux from THP-1 cells. However, pre-.beta.1-HDL-deficient plasma reconstituted with 16% pre-.beta.1-HDL was more active than whole plasma, demonstrating that pre-.beta.1-HDL does promote cholesterol efflux actively. The amt. of cellular cholesterol present in reisolated pre-.beta.1-HDL was 1.5- to 2-fold greater after incubation of the cells with whole plasma than after incubation of the cells with pre-.beta.1-HDL-deficient plasma or plasma treated with the anti-pre-.beta.1-HDL antibody. However, the anti-pre-.beta.1-HDL antibody did not inhibit cholesterol efflux. We conclude that whereas pre-.beta.1-HDL may be the first product of apolipoprotein A-I lipidation during the formation of HDL but may not play a major role in transferring cellular cholesterol to HDL.

CC 9-16 (Biochemical Methods)
Section cross-reference(s): 13

ST beta HDL cholesterol efflux

IT Apolipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(A-I; delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT Animal cell line
(THP-1; delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT Blood plasma
Fibroblast
Human
Macrophage
(delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT Biological transport
(efflux; delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(high-d., pre .beta.1-; delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT Antibodies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(monoclonal; delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT Gel electrophoresis
(two-dimensional; delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

IT 57-88-5, Cholesterol, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(delineation of role of pre-.beta.1-HDL in cholesterol efflux using isolated pre-.beta.1-HDL)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:14791 HCAPLUS
DOCUMENT NUMBER: 134:159762
TITLE: A new sandwich enzyme immunoassay for measurement of plasma pre-.beta.1-HDL levels
AUTHOR(S): **Miyazaki, Osamu**; Kobayashi, Junji; **Fukamachi, Isamu**; Miida, Takashi; Bujo, Hideaki; Saito, Yasushi
CORPORATE SOURCE: Daiichi Pure Chemicals Company, Tokyo, Japan
SOURCE: Journal of Lipid Research (2000), 41(12), 2083-2088
CODEN: JLPRAW; ISSN: 0022-2275
PUBLISHER: Lipid Research, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Pre-.beta.1-HDL, a putative discoid-shaped high d. lipoprotein (HDL) of approx. 67-kDa mass that migrates with pre-.beta. mobility in agarose gel electrophoresis, contains apolipoprotein A-I (apoA-I), phospholipids, and unesterified cholesterol. It participates in the retrieval of cholesterol from peripheral tissues. In this study we established a new sandwich enzyme immunoassay (EIA) for measuring plasma pre-.beta.1-HDL using mouse anti-human pre-.beta.1-HDL monoclonal antibody (MAb 55201) and goat anti-human apoA-I polyclonal antibody. MAb 55201 reacted with apoA-I in lipoprotein [A-I] with mol. mass less than 67 kDa, and with pre-.beta.1-HDL sepd. by nondenaturing two-dimensional electrophoresis, whereas it did not react with apoA-I in .alpha.-HDL. Pre-.beta.1-HDL levels measured by this method declined when incubated at 37.degree.C for 2 h, whereas this decrease was not obsd. in the presence of 2 mM lecithin:cholesterol acyltransferase inhibitor 5,5'-dithiobis (2-nitrobenzoic acid). To clarify the clin. significance of measuring pre-.beta.1-HDL by this method, 47 hyperlipidemic subjects [male/female 22/25; age 55 .+-. 14 yr; body mass index 25 .+-. 4.5 kg/m2; total cholesterol (TC) 245 .+-. 64 mg/dL; triglyceride (TG) 232 .+-. 280 mg/dL; HDL cholesterol (HDL-C) 51 .+-. 23 mg/dL] and 25 volunteers (male/female 15/10; age 36 .+-. 9.3 yr; body mass index 23 .+-. 3.5 kg/m2; TC 183 .+-. 28 mg/dL; TG 80 .+-. 34 mg/dL; HDL-C 62 .+-. 15 mg/dL) were involved. Plasma pre-.beta.1-HDL levels were significantly higher in hyperlipidemic subjects than in volunteers (39.3 .+-. 10.1 vs. 22.5 .+-. 7.5 mg/mL, $P < 0.001$) whereas plasma apoA-I levels did not differ (144.2 .+-. 28.4 vs. 145.3 .+-. 16.3 mg/dL). These results indicate that this sandwich EIA method specifically recognizes apoA-I assocd. with pre-.beta.1-HDL.

CC 9-10 (Biochemical Methods)
Section cross-reference(s): 14
ST high density lipoprotein detn enzyme immunoassay
IT Apolipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(A-I, monoclonal antibodies to; plasma .beta.1-HDL levels detn. by sandwich immunoassay using monoclonal antibody against apoA-1)
IT Immunoassay
(enzyme; plasma .beta.1-HDL levels detn. by sandwich immunoassay using monoclonal antibody against apoA-1)
IT Lipoproteins
RL: ANT (Analyte); ANST (Analytical study)
(high-d., pre-.beta.1; plasma .beta.1-HDL levels detn. by sandwich immunoassay using monoclonal antibody against apoA-1)
IT Lipids, analysis
RL: ANT (Analyte); ANST (Analytical study)
(hyperlipidemia; plasma .beta.1-HDL levels detn. by sandwich immunoassay using monoclonal antibody against apoA-1)
IT Antibodies

RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(monoclonal; plasma .beta.1-HDL levels detn. by sandwich immunoassay using monoclonal antibody against apoA-1)

IT Blood analysis
(plasma .beta.1-HDL levels detn. by sandwich immunoassay using monoclonal antibody against apoA-1)

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:441927 HCAPLUS
DOCUMENT NUMBER: 133:72947
TITLE: Monoclonal antibody against apolipoprotein A-I
INVENTOR(S): **Miyazaki, Osamu; Fukamachi, Isamu**
PATENT ASSIGNEE(S): Daiichi Pure Chemicals Co., Ltd., Japan
SOURCE: PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000037632	A1	20000629	WO 1999-JP7106	19991217
W: AU, CA, US				
RW: DE, FR, GB				
CA 2356738	AA	20000629	CA 1999-2356738	19991217
JP 2000239300	A2	20000905	JP 1999-358548	19991217
EP 1142996	A1	20011010	EP 1999-959883	19991217
R: DE, FR, GB				

PRIORITY APPLN. INFO.: JP 1998-364295 A 19981222
WO 1999-JP7106 W 19991217

AB A monoclonal antibody reacting specifically with (1) apoA-I having a mol. wt. of not more than 150,000 and occurring in HDL free from apoA-II; and (2) apoA-I not binding to a lipid; a hybridoma producing this antibody; a method of immunol. assaying apoA-I characterized by reacting the antibody with a specimen; and an assay reagent for apoA-I which contains the antibody. The specific apoA-I thus assayed is usable as a novel indication of lipid metabolic error, etc.

IC ICM C12N015-08
ICS C07K016-18; C12N005-12; C12P021-08; G01N033-53; G01N033-577
CC 15-3 (Immunochemistry)
ST lipid metabolic disorder apoA monoclonal antibody
IT Apolipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(A-I; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)
IT Immunoassay
(enzyme; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)
IT Lipoproteins
RL: AMX (Analytical matrix); ANST (Analytical study)
(high-d.; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)
IT Lipids, biological studies
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(hyperlipidemia; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

IT Lipids, biological studies
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(metabolic disorders; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

IT Hybridoma
Immunoassay
(monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

IT Lipids, analysis
RL: AMX (Analytical matrix); ANST (Analytical study)
(monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

IT Antibodies
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

IT Immunoassay
(radioimmunoassay; monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

IT 12786-37-7, Lipoprotein A-II (human blood plasma high-density protein moiety)
RL: REM (Removal or disposal); PROC (Process)
(monoclonal antibody against apolipoprotein A-I for diagnosis of lipid metabolic disease)

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT